Table 1 | A comparison of HD patients with and without pain

	No pain (n=75)		Chronic pain (n=130)		ANOVA
	Mean	s.d.	Mean	s.d.	P
Maintenance HD (months)	36.1	43.3	64.9	67.8	**0.0011**
Age (years)	62.7	14.1	58.9	13.6	0.0598
24 h diuresis (l)	0.5	0.5	0.3	0.5	**0.0065**
BMI	25.0	4.1	25.4	4.2	0.4855
SBP before HD (mm Hg)	136.4	26.5	133.8	24.7	0.4797
DBP before HD (mm Hg)	77.7	11.0	76.0	11.2	0.3014
SBP after HD (mm Hg)	131.3	25.7	126.6	26.4	0.2175
DBP after HD (mm Hg)	75.5	11.9	73.8	12.3	0.3368
Total protein (g/l)	6.9	0.6	7.0	0.6	0.1406
Albumin (g/l)	3.6	0.4	3.7	0.5	0.1526
Hemoglobin (g/l)	10.6	1.6	11.1	2.8	0.2135
CaxP	47.2	17.1	53.1	17.3	**0.0190**
PTH	215.6	181.6	467.0	595.9	**0.0004**
kt/V	1.1	0.2	1.2	0.3	0.1710
kt/V weekly	3.4	0.6	3.5	0.6	0.1543
Mean UF	2610.7	857.9	2706.9	844.7	0.4354
CRP (g/l)	12.1	15.0	14.2	23.7	0.4990
HADS-A	4.3	3.4	6.5	3.4	**0.0000**
HADS-D	4.6	4.0	6.8	4.1	**0.0002**
MMSE	25.6	3.1	27.2	7.5	0.2559
SF-36v2					
GH	49.1	19.6	38.4	17.7	**0.0001**
BP	86.8	22.0	44.5	25.6	**0.0000**
PF	52.6	31.7	36.6	29.8	**0.0004**
RP	57.4	44.3	40.5	43.1	**0.0088**
RE	70.8	42.6	58.5	45.4	0.0624
VT	59.2	22.4	44.1	19.7	**0.0000**
MH	69.9	19.2	59.2	20.6	**0.0003**
SF	77.4	26.0	59.5	28.4	**0.0000**

Abbreviations: ANOVA, analysis of variance; BMI, body mass index; BP, bodily pain; CaxP, calcium-phosphorus ion product; CRP, C-reactive protein; DBP, diastolic blood pressure; GH, general health; HADS, Hospital Anxiety and Depression Scale; HD, hemodialysis; MH, mental health; MMSE, Mini-Mental State Examination; PF, physical functioning; PTH, parathyroid hormone; RE, role-emotional limitation; RP, role-physical limitation; SBP, systolic blood pressure; SF, social functioning; SF-36v2, Version 2.0 of Short Form 36; UF, ultrafiltration; VT, vitality.
The bold values indicate statistically significant difference ($P<0.05$).

depression was already confirmed.[2] Moreover, it may predispose patients to consider withdrawal of dialysis. We performed a cross-sectional study ($n=205$) evaluating the prevalence of pain among a cohort of long-term HD patients and compared depression (Hospital Anxiety and Depression Scale (HADS)) and quality of life (Version 2.0 of Short Form 36 (SF-36)) in patients with and without chronic pain.[3] The results are summarized in Table 1. Significant differences were found in anxiety (HADS-A), depression (HADS-D), and all SF-36 scales. Patients with pain showed higher levels of calcium-phosphorus ion product and parathyroid hormone than those without pain. Perhaps those with pain and depression are more non-adherent to dietary and fluid restriction, which in turn may lead to worse outcomes in the future.[4] Data on pain prevalence or even the subanalysis in the study by the Duarte group would be interesting. Some data about pain are in KDQOL-SF (body pain).

While investigating the problem of how to create coping strategies to deal with dialysis treatment, depression, and pain, we found in our study some differences between patients who were on the waiting list for kidney transplantation and those who were not on the list (Trafidło *et al.*; www.abstracts2view.com/wcn). HD patients with chronic pain evidenced that being on the waiting list was associated with diminished cognitive/behavioral scores in the Pain Coping Strategies Questionnaire (less catastrophizing; $P=0.009$). A similar comparison addressing depression-coping strategies would be important.

Finally, we appreciate Duarte *et al.* for the first randomized trial on CBT in the HD population and also encourage nephrologists to pay more attention to diagnosing and treating depression in HD patients because it may be essential in pain management.

1. Duarte PS, Miyazaki MC, Blay SL *et al.* Cognitive-behavioral group therapy is an effective treatment for major depression in hemodialysis patients. *Kidney Int* 2009; **76**: 414–421.
2. Davison SN, Jhangri GS. The impact of chronic pain on depression, sleep, and the desire to withdraw from dialysis in hemodialysis patients. *J Pain Symptom Manage* 2005; **30**: 465–473.
3. Trafidlo E, Kusztal M, Weyde W *et al.* Depression, mental functioning and quality of life among haemodialyzed patients with pain. *NDT Plus* 2008; **1**(Suppl 2): ii186–187.
4. Cukor D, Peterson RA, Cohen SD *et al.* Depression in end-stage renal disease hemodialysis patients. *Nat Clin Pract Nephrol* 2006; **2**: 678–687.

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The Authors Reply: We appreciate the interest in our recently published article 'Cognitive-behavioral group therapy is an effective treatment for major depression in hemodialysis (HD) patients', which presented the advantages of a specific psychological approach to treat depression in Brazilian patients undergoing hemodialysis.[1,2] The authors recognize the importance of studying in more depth the relationship of physical symptoms, such as pain, in chronic kidney disease and its treatment, with both the functional status and the psychosocial well-being of these patients.

The prevalence of chronic pain in patients of our study was high. At the beginning of the study, the percentage of patients with body pain was similar in the two study groups: 84.8% ($n=39$) in the cognitive-behavioral therapy group and 84.1% ($n=37$) in the control group. After 3 months, this prevalence rate decreased in the intervention group (78.0% ($n=32$)) and increased in the control group (88.6% ($n=39$)). Muscle pain was present in 71.7% ($n=33$) and 72.7% ($n=32$) of the patients in the intervention and the control groups, respectively, at baseline. The corresponding figures after 3 months of study were 73.2 and 72.7%, respectively. Seventy-two percent ($n=33$) of the patients in the intervention group and 72.7% ($n=32$) of the patients in the control group stated at baseline that the pain interfered

with their normal work. After 3 months, these percentages changed to 58.5 and 81.8% in the cognitive-behavioral therapy (CBT) and control groups, respectively.

The relationship between pain and depression in end-stage renal disease patients is important. The impact of CBT in alleviating body pain in these patients requires future research.

1. Kusztal M, Trafidło A, Weyde W *et al.* Cognitive-behavioral group therapy is an effective treatment for major depression in hemodialysis (HD) patients. *Kidney Int* 2010; **77**: 646–647.
2. Duarte PS, Miyazaki MC, Blay SL *et al.* Cognitive-behavioral group therapy is an effective treatment for major depression in hemodialysis patients. *Kidney Int* 2009; **76**: 414–421.

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Sudden cardiac death and mineral metabolism in chronic kidney disease

Pun *et al*. showed that reductions in the estimated glomerular filtration rate (eGFR) were associated with an increase in the risk of sudden cardiac death in a graded fashion in patients with coronary artery disease. The authors claimed that decreased eGFR induces many metabolic and physiological changes that might be responsible for increased sudden cardiac death in chronic renal failure patients.[1] Although the study is informative, we are especially concerned about the relationship between calcium, phosphorus, Ca × P product, parathyroid hormone levels, and sudden cardiac death in the study population. It is well established that abnormalities in mineral metabolism are apparent early in the course of chronic kidney disease (CKD). Beginning in CKD stage 3, the ability of the kidneys to appropriately excrete a phosphate load is diminished, leading to hyperphosphatemia, elevated parathyroid hormone, and decreased vitamin D levels. Furthermore, there is emerging evidence linking some of these abnormalities (for example, hyperphosphatemia and hypercalcemia) to the high cardiovascular morbidity and mortality experienced by nondialyzed patients with CKD. One of the mechanisms for deranged mineral metabolism to induce cardiovascular disorders is thought to be the calcification of the vascular tree that result in arterial stiffness. Arterial stiffness of the large arteries has important clinical consequences: raised systolic blood pressure, increased pulse pressure, left ventricular hypertrophy, and reduced coronary perfusion.[2,3]

Experimental evidence showed that high levels of phosphate and/or calcium directly activated genes related to an osteoblastic phenotype in the smooth muscle cells.[4] In addition, elevated phosphorus and calcium stimulated the transformation of vascular smooth muscle cells into osteoblast-like cells *in vitro* using cell-culture techniques.[5] Besides, clinical evidence also suggests that high pre-dialysis serum phosphate is a powerful predictor of sudden cardiac death.[6]

Since the regular control of calcium, phosphorus, and parathyroid hormone in chronic renal failure patients is strongly recommended, if available, the presently informative results by Pun *et al*. would have been much more valuable with the addition of parameters of mineral metabolism in the adjusted analyses.

1. Pun PH, Smarz TR, Honeycutt EF *et al.* Chronic kidney disease is associated with increased risk of sudden cardiac death among patients with coronary artery disease. *Kidney Int* 2009; **76**: 652–658.
2. Clinical Practice Guideline for the Diagnosis, Evaluation, Prevention, and Treatment of Chronic Kidney Disease–Mineral and Bone Disorder (CKD-MBD). *Kidney Int* 2009; **76**(Suppl 113): S3–S8.
3. Covic A, Gusbeth-Tatomir P, Goldsmith DJ. Arterial stiffness in renal patients: an update. *Am J Kidney Dis* 2005; **45**: 965–977.
4. Cozzolino M, Brancaccio D, Gallieni M *et al.* Pathogenesis of vascular calcification in chronic kidney disease. *Kidney Int* 2005; **68**: 429–436.
5. Clinical Practice Guideline for the Diagnosis, Evaluation, Prevention, and Treatment of Chronic Kidney Disease–Mineral and Bone Disorder (CKD-MBD). *Kidney Int* 2009; **76**(Suppl 113): S22–S49.
6. Ganesh SK, Stack AG, Levin NW *et al.* Association of elevated serum PO(4), Ca x PO(4) product, and parathyroid hormone with cardiac mortality risk in chronic hemodialysis patients. *J Am Soc Nephrol* 2001; **12**: 2131–2138.

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The Authors Reply: We appreciate Drs Afsar and Elsurer for their interest and comments about our study.[1] We agree that disordered mineral metabolism has been associated with cardiac risk in hemodialysis patients,[2] in those with less severe chronic kidney disease,[3] and in some patients who lack overt kidney disease.[4] We now report available laboratory data on serum calcium, phosphorus, and parathyroid hormone (PTH) concentrations obtained within 3 months prior to cardiac catheterization in the study cohort. Concurrent PTH data were unavailable on the majority of patients, but calcium and phosphorus data were available in 46% of patients with glomerular filtration rate (GFR) <15 and in 18% of patients with GFR $\geqslant 15$ (Table 1). Calcium and calcium–phosphorus product had no significant relationship with the composite outcome, but phosphorus had a significant relationship (hazard ratio 1.27, 95% confidence interval 1.04–1.55) in univariate analysis. However, this relationship was abolished after accounting for baseline GFR. Accounting for serum phosphorus did not alter the relationship between GFR and outcome in an adjusted model. Therefore, our study findings could not be explained by measured abnormalities in mineral metabolism, although the analysis was limited by missing data.